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Are Volcanoes Hazardous to Your Health? What Have We Learned From Mount St Helens?

"IS THE VOLCANIC ASH harmful to health?" This was the question posed repeatedly to physicians and public health officials when the violent eruption of Mount St Helens on May 18, 1980, and smaller eruptions later in the summer showered ash over a considerable portion of central, eastern and southwestern Washington state and northwestern Oregon state. The purpose of this review is to report what is known about the health effects of volcanic ash, point out the deficiencies in our knowledge and describe the ways in which the health effects of this unusual form of environmental pollution are being evaluated.

The May 18, 1980, eruption of Mount St Helens was the first major volcanic eruption in the 48 contiguous states of the United States since the eruptions of Lassen Peak in California between 1914 and 1917. As volcanic eruptions go, the eruption of Mount St Helens was quite modest, with only about 2.7 cu km of volcanic rock displaced, compared with eruptions in ancient times that displaced up to 1,000 cu km of material.[1] In the past century, however, only the eruptions of Santa Maria in Guatemala (1902), Krakatoa in Indonesia (1883) and Mount Katmai in Alaska (1912) have surpassed Mount St Helens in magnitude. In terms of energy released, the May 18 eruption was on the order of 100 times the generating capacity of all US electric power stations.[1] Put in another way, the sustained power output of the May 18 eruption may be compared with the serial detonation of 27,000 bombs the size of the one dropped on Hiroshima—nearly one per second for nine hours.

With this amount of energy output, it is not surprising that volcanoes can kill. Probably the earliest report of death from a volcano is to be found in the letters of Pliny the Younger, writing to the historian Tacitus about the death of his father, Pliny the Elder, who succumbed in the eruption of Mount Vesuvius in AD 79.[2] In the 20th century alone, the cumulative mortality from volcanic eruptions is in the region of 55,000, roughly 36,000 of these deaths attributable to the eruption of Mount Pelée in 1902 which caused the destruction of the town of St. Pierre on North Martinique in the French West Indies, and massive flooding and tidal waves.[3] The death toll from the eruption of Mount St Helens was small in comparison, with 62 persons killed or missing. However, this figure would have been considerably higher if the eruption had not occurred on a Sunday when loggers were not on the mountain and if Governor Ray and the US Forest Service had not insisted on keeping the area closed to the public despite vociferous demands for freedom of access.

Causes of Death From the May 18 Eruption

Eisele and co-workers[4] recently reported the autopsy findings in the first 25 bodies recovered after the eruption. The most common cause of death was asphyxia due to inhalation of volcanic

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ABBREVIATIONS USED IN TEXT

CDC = Centers for Disease Control
EPA = Environmental Protection Agency
OSHA = Occupational Safety and Health Administration
SiO_2 = silicon dioxide
TSP = total suspended particulates

ash; 17 of the deaths were attributed to asphyxia and it was thought to be a contributory factor in two additional deaths. The asphyxia was caused by ash that had mixed with mucus to form occlusive plugs in the upper airways. Two of the victims managed to walk off the mountain and subsequently died in hospital. At autopsy they were found to have a purulent tracheobronchitis that was attributed to inhalation of volcanic gas and particulate matter. Interestingly, a companion who walked off the mountain with these two and was admitted to hospital with them, survived and was eventually discharged. This raises interesting questions about why he should have survived when his companions, who presumably received an approximately equal dose of hot volcanic gases and inhaled volcanic ash, did not.

Thermal burns were directly responsible for some deaths, being a major cause of death in three persons and contributory in two more. In contrast to the persons who died as a result of thermal burns, there was no substantial premortem burning in the victims who died of asphyxia and physical injury. In these victims, there were, however, postmortem thermal changes, with the skin showing a tanning or mummification effect, being leathery and darkly discolored. The portions of bodies that were found buried in ash showed skeletonization and disarticulation, which appeared to be postmortem changes.

This account of the immediate cause of death in the victims of the Mount St Helens eruption is in agreement with the analysis by Thorarinsson[5] of the large number of deaths resulting from the eruption of Mount Vesuvius in AD 79. It seems likely that suffocation from ash is the most immediate hazard and that the accompanying gases, such as sulfur dioxide, though present and noticeable, do not occur in toxic concentrations.

Health Effects of the May 18 Ashfall

Immediately following the May 18 eruption, the process of identifying the composition of the ash began[6,7] and an epidemiologic team from the Centers for Disease Control (CDC) arrived to assist the Washington State Department of Social and Health Services to evaluate the acute and potential long-term health effects.[8] This involved setting up a surveillance network that included 18 main hospitals in the ashfall area of central and eastern Washington, with nine hospitals in the affected area of the state being added after the May 25 eruption. Hospitals were contacted daily by telephone for total numbers of emergency room visits and other admissions for respiratory and other illnesses.

For the week after the May 18 eruption and for the following two weeks there were appreciable increases in emergency room visits and hospital admissions for respiratory complaints in the areas of the heaviest ashfall (Table 1). After the

TABLE 1.—*Emergency Room (ER) Visits and Admissions for Respiratory Diseases for Washington Hospitals, May-June 1980**

Location (No. of Hospitals)	*Ashfall, mm*	*ER Visits/Admissions*				
		May 11-17	*May 18-24*	*May 25-31*	*June 1-7*	*June 8-14*
Eastern Washington						
Ritzville, Moses Lake and Othello (3)	30-70	14/12	72/35	31/12	14/8	14/8
Yakima (2)	8	49/15	150/33	99/19	101/21	102/16
Pullman, Soap Lake, Ellensburg and Ephrata (4)	5-10	14/6	24/7	15/14	18/8	13/10
Spokane (2)	3-4	15/9	55/17	61/14	45/9	36/16
TOTAL		92/42	301/92	206/59	178/46	165/50
Western Washington						
Centralia and Chehalis (2)	8-10	19/6	31/7	62/9	33/5	27/6
Longview (2)	1-2	62/31	54/14	85/25	51/16	53/16
Aberdeen (2)	0.3-0.5	76/10	77/12	95/19	71/20	80/14
McCleary and Shelton (2)	Trace	19/6	11/4	20/6	11/4	10/7
TOTAL		176/53	173/37	262/59	166/45	170/43

*From Baxter et al.[8]

May 25 eruption, there was an increase in the affected areas in emergency room visits, but not in hospital admissions, for respiratory complaints. The main reasons for the increase in emergency room visits seem to have been airways-related problems, such as bronchitis and exacerbations of asthma.[8]

Although respiratory problems were the most common diagnoses, there was also an appreciable increase in the number of visits for eye irritation and abrasion, foreign bodies and conjunctivitis during the first two weeks after the eruption.[8]

These data collected by the CDC and the Washington State Department of Social and Health Services certainly bear out the clinical experience of many of the physicians in practice in the affected areas. However, it would be a mistake to place too much faith in the actual numbers because the disruption of normal life was so great, with travel very hazardous and many physicians' offices closed, that it is hard to know whether the numbers obtained were in fact an underestimate of the real extent of the problem or an overestimate.

An Educated Guess About the Health Hazard

In evaluating the potential risk of an inhaled particulate, the crucial questions that must be answered relate to the size range and composition of the inhaled material, and to the total amount inhaled. The size range is important because this determines the site of deposition in the airways and alveoli. The composition and total amount inhaled are important because they influence the way in which the lungs' defenses cope with the invader.

First is the question of the size range. The first published report of the particle size distribution of the ash was that of Fruchter and co-workers[6] who showed that the particle size distribution of the fallen ash was complex and related to the distance from the volcano. Of particular importance in relation to the effect on the lungs was the finding that between 94 percent and 99 percent of the particles, by count, were within the respirable range (less than 10 μm) in samples taken at different sites across Washington state. There was little doubt, therefore, that most of the ash particles were small enough to penetrate into the alveoli.

Second is the composition of the ash. Because of the critical importance of this question, it was addressed by countless numbers of investigators, working singly in the solitude of their basements or in large laboratories with sophisticated facilities. The major concern related to the free silica content. The fallen ash had a dacitic (high silica) composition. However, most of the silica was in the form of silicates,[6] which are generally not thought to have much fibrogenic potential. The form of silica with the greatest fibrogenic potential is free or crystalline silica (silicon dioxide, SiO_2), which exists in three polymorphs, quartz, tridymite and cristobalite, listed in increasing order of fibrogenicity. The crystalline silica content of the Mount St Helens ash proved to be quite difficult to quantitate because of considerable analytical problems in separating the crystalline silica from the silicates. Initial estimates of the crystalline silica content ranged from less than 3 percent[6] to about 20 percent. This controversy was eventually resolved by a round robin organized by the National Institute for Occupational Safety and Health, Morgantown, West Virginia, in which approximately 25 laboratories analyzed identical samples in a standardized way and using their own methods.[7,8] The consensus that developed from this round robin was that the Mount St Helens ash contained levels of crystalline silica in the range of 3 percent to 7 percent, as part quartz and part cristobalite.[8] No fibrous minerals were found.

Other questions of importance relating to the composition of the ash were the radionuclide and fluoride content. The answers were readily produced and were reassuring: the radionuclide content of the ash was comparable to that of ordinary soil and the leachable fluoride and other heavy metal concentrations were not increased.[6,7]

The third key question in evaluating the potential health effects of the ash related to the inhaled dose. Table 2 shows the average concentrations of respirable dust and the range of dust concentrations measured by the National Institute of Occupational Safety and Health industrial hygienists in a variety of occupational settings in June 1980. To put these in perspective they should be compared with existing criteria for inhaled particulates and silica. The relevant criteria are therefore the ambient air quality standards of the Environmental Protection Agency (EPA) for particulates[9] and the National Institute of Occupational Safety and Health recommendations for occupational environmental exposure limits for crystalline silica.[10]

The EPA standards for particulates are as follows: primary air quality standard, 0.260 mg per

cu m; air pollution alert, 0.375 mg per cu m; warning, 0.625 mg per cu m; emergency level, 0.875 mg per cu m, and significant harm level, 1.0 mg per cu m. It is important to point out that these standards are set for industrial particulate emissions, which are usually associated with other airborne pollutants such as sulfur dioxide and the oxides of nitrogen. It is therefore not clear whether these standards are at all relevant to airborne volcanic ash, especially in a rural setting where the levels of other airborne pollutants are probably negligible.

Rather than using the EPA's ambient air quality standards for particulates, a reasonable argument might be made for using the standards of the American Conference of Governmental Industrial Hygienists for nuisance dust,[11] as the volcanic dust does not constitute an industrial emission. These standards set the permissible respirable dust level at 5 mg per cu m time weighted average, with a total dust level of 10 mg per cu m time weighted average. This means that the permissible dust level over a given period—for example, a work shift—must have an average of less than 5 mg per cu m for respirable dust and less than 10 mg per cu m for total dust. These standards are used in a number of dusty occupations, for example, that of grain workers. However, they are not applicable to volcanic dust inasmuch as one of the important criteria of a nuisance dust is that it is not capable of causing pulmonary fibrosis. Because the crystalline silica content of the ash of 3 percent to 7 percent makes the volcanic dust potentially fibrogenic, it is clearly not in the nuisance dust category.

The occupational and environmental standards for crystalline silica have been set by the Occupational Safety and Health Administration (OSHA) and occupational environmental exposure limits recommended by the National Institute of Occupational Safety and Health.[10] The OSHA permissible exposure level is calculated from the formula

$$\frac{10}{\%\ \text{respirable free}\ SiO_2 + 2}$$

mg per cu m. For example, assuming the volcanic dust contains 6 percent respirable crystalline silica, the OSHA standards would therefore permit exposure over a working lifetime to dust levels of 1.25 mg per cu m. The National Institute of the Occupational Safety and Health recommendation for an occupational exposure limit for crystalline silica is 50 μg per cu m time weighted average. These levels would be reached by exposure to respirable dust concentrations of just above 0.8 mg per cu m with a 6 percent crystalline

TABLE 2.—*Total and Respirable Dust Levels for Different Occupations, June 1980**

Job	*Total Dust mg/cu m mean (range)*	*Respirable Dust† mg/cu m mean (range)*
Cleanup Crews		
Hand-shoveling and sweeping	2.65(0.64-6.46)	0.46(0.02-2.08)
Sweeper-truck or broom-truck drivers	5.50(0.60-23.1)	0.64(0.03-2.83)
Front-end-loader operators	...(6.17-63.6)	0.50(0.21-0.96)
Grader operators	5.96(0.01-31.9)	0.56(0.01-2.33)
Water-truck drivers	1.48(0.23-6.14)	0.21(0.04-0.64)
Truck drivers	...	0.19(0.10-0.37)
Manual hosing	...	0.05(0.03-0.06)
Rubbish Worker	9.01(0.73-25.5)	0.67(0.11-5.51)
Idaho Forest Worker	...	0.48(0.01-2.46)
Agricultural Worker	1.42(0.79-3.20)	0.44(0.01-1.39)
Law Enforcement Personnel	0.57(0.04-4.17)	0.10(0.01-0.23)
Area Samples		
Homes	0.09(0.03-0.20)	0.03(0.01-0.08)
Schools	0.30(0.20-0.50)	0.06(0.01-0.11)
Commercial establishments	0.30(0.11-0.44)	0.09(0.03-0.20)
Autos	...	0.10
Combines		
With air-conditioned cabs	5.82(4.24-8.20)	0.40(0.17-0.74)
Without air-conditioned cabs	...	2.24(2.20-2.38)
Trucks	2.10(1.70-2.75)	0.35(0.21-0.50)

*Data obtained by National Institute of Occupational Safety and Health industrial hygienists as reported in the Centers for Disease Control's Mount St Helens Volcano Reports Nos. 12 and 17.[7]

†Respirable dust particles collected on a 37 μm polyvinyl chloride filter after passing through a 10 μm cyclone.

silica content. At these levels, according to the epidemiologic data available at the time the standards were set, silicosis would not be expected to develop in an occupational setting over the course of a working lifetime. However, more recent epidemiologic data[12] obtained from the Vermont quarry workers on whom the original standards are based[13-16] cast doubt on the validity of the original longitudinal data, some of which may have been flawed by a leaky spirometer. The environmental and occupational standards for silica exposure therefore probably need to be reexamined in the light of the new data.

How do these standards relate to the environmental conditions in the ashfall areas during the spring and summer of 1980 and to occupational exposure during the cleanup phase following the eruptions (shown in Table 2) and the exposures received by the loggers involved in the salvage operations of the lumber on Mount St Helens? The occupations that had an average respirable dust concentration of 0.45 mg per cu m or more exceeded the National Institute of Occupational Safety and Health recommended exposure limits (assuming a crystalline silica level of 6 percent) of 0.8 mg per cu m 15 percent to 31 percent of the time. If exposures at this level were continued for many years and were not reduced by the use of respiratory protection, these workers would be expected to be at increased risk of silicosis developing.

These occupational data were obtained in 1980. Very few occupational groups have had continued exposure to appreciable dust concentrations since the cleanup was completed in the late summer of 1980. Exceptions to this are the loggers who have continued to work on the salvage operations, retrieving the fallen timber off the mountain, and agricultural workers in eastern Washington.

Compared with some of the occupational exposures, the environmental exposures were modest. Peak levels of total suspended particulates (TSP) were indeed very high in some areas, especially in the path of the plume where TSP levels in excess of 30 mg per cu m were recorded.[7] Because the Environmental Protection Agency's primary air quality TSP standard is 0.26 mg per cu m, with the significant harm level set at 1 mg per cu m for a 24-hour exposure, such high TSP levels would be very likely to provoke acute respiratory tract illness in exposed, susceptible persons or exacerbate preexisting chronic respiratory problems. Fortunately, however, the high levels did not persist, though the TSP levels in the ashfall areas were mostly higher than normal throughout the summer of 1980, as noted by Peter Baxter, MD, and Roy Ing, MD, of the Chronic Diseases Division, Center for Environmental Health, Centers for Disease Control, Atlanta (personal memorandum, May 3, 1981). This led on a number of occasions to air pollution alerts, when the TSP level exceeded 0.375 mg per cu m.

The very low levels of respirable dust measured in homes and other buildings (Table 2) were reassuring and suggest that the general population did not receive and is not receiving a dose of inhaled ash high enough to cause concern. The relatively low levels of dust measured indoors also lent some credibility to the recommendations that persons with heart and lung disease should stay indoors during and after ashfalls.

How the Lungs Handle Particulates

Part of the process of making an educated guess about the potential health hazard of the volcanic ash must involve evaluating the ways in which the lungs handle particulates in general and, more specifically, how the lungs' defenses cope with particulates containing crystalline silica. The deposition of aerosols in the respiratory tract has been the subject of an excellent recent review by Brain and Valberg[17] and a two-part monograph.[18]

Because the respiratory tract is in direct communication with our environment, it is not surprising that it has elaborate defense mechanisms. For the purpose of describing deposition of aerosols, it is usual to divide the respiratory tract into three anatomically distinct compartments; nasopharyngeal, tracheobronchial and pulmonary. The first line of defense an inhaled particle meets is the nasopharynx where turbulent flow through the narrow passages of the nose enhances inertial deposition. Particles that succeed in penetrating the tracheobronchial compartment, which extends to the level of the terminal bronchioles (0.7 mm in diameter), meet an asymmetric system of dichotomously branching tubes, which encourages inertial impaction of particles at bifurcations and settling of particles on the surface of the airways. The pulmonary compartment includes the respiratory bronchioles, alveolar ducts and alveoli, with a huge surface area but small air spaces. Because the bulk movement of air is very slow in this compartment, sedimentation and diffusion can occur.

As noted above, over 94 percent of the volcanic

ash particles by count were in the respirable range, less than 10 μm. The amount and site of deposition in the lungs are most closely linked to median aerodynamic size. For the particles in the 1 to 10 μm size range, inertial impaction and settling are the major mechanisms of deposition and deposition mainly occurs in the nasopharyngeal and tracheobronchial compartments from which the particles are readily removed by the mucociliary escalator. Very small particles, 0.1 μm or smaller, penetrate the pulmonary compartment and tend to be retained very efficiently; they then take considerably longer to be cleared. Particles in the intermediate size range of 0.1 to 1 μm tend to be retained less avidly in the pulmonary compartment than the smaller particles but, if deposited, are also cleared relatively slowly.

It is important to appreciate that there are large differences between persons in the site of deposition and the amount of an aerosol that is deposited. The deposition is influenced by a number of factors such as the size, shape and density of the particles, respiratory anatomy, breathing pattern, dead space volume and tidal volume. Disease, including smoking-related inflammatory changes, will also affect the deposition. The effect of smoking is particularly important, as shown recently by Cohen and colleagues[19] who demonstrated that the long-term clearance of a magnetic dust (iron oxide) was much slower in smokers than in nonsmokers. In their study, after 11 months the smokers retained about five times more iron oxide dust in their lungs than did the nonsmokers. The implication of these data in terms of the volcanic dust is very clear, namely, that any dust particles small enough to penetrate the pulmonary compartment will stay around longer in smokers than in nonsmokers.

If the dust particles were inert, sizable differences between persons in clearance rates would be relatively unimportant. However, because the dust contains crystalline silica, one could predict that deposition in the respiratory tract will provoke a response from the alveolar macrophages, the main scavenger cells of the pulmonary compartment. Macrophages have an important role in influencing the length of time inhaled insoluble particles are retained in the respiratory tract. If particles that are deposited on airway and alveolar surfaces are not phagocytized, they are more likely to pass through or between epithelial cells to the connective tissue and lymph nodes beneath the epithelium where they are retained longer and therefore have a greater opportunity to cause disease. However, the phagocytic role of macrophages can be compromised by a large number of agents, including crystalline silica.[17,18,20,23] The reason for the cytotoxic effect of crystalline silica is not yet clear. What is important is that the dead or dying macrophages release substances that can attract fibroblasts and elicit fibrogenic responses. Thus, even though a macrophage is a very effective first line of defense in protecting the alveolar surface, it has the potential of injuring the lung. This represents an excellent example of biological backfire in that the very mechanism designed to protect the lungs now becomes the mechanism promoting lung injury.

From this brief review of the lung defense mechanisms most clearly relevant to particulates, we can probably speculate that volcanic ash may cause both acute and chronic respiratory tract problems, the acute problems being irritation of the nasopharynx and airways resulting in hypersecretion of mucus, sore throat and cough, and a bronchoconstrictor response in susceptible persons, with the ash acting as a nonspecific, nonallergenic stimulus. In otherwise healthy persons, these acute effects would be expected to pose a relatively minor health problem. In persons with preexisting lung disease, however, one might predict that the additional insult and burden on the already overloaded or impaired lung defenses might constitute a serious risk. Likewise, in persons with reactive airway disease, the precipitation of an acute attack of asthma or the exacerbation of existing bronchoconstriction may have serious consequences.

The main chronic respiratory tract problem of concern in persons exposed over many years to dangerous levels of inhaled volcanic ash is silicosis, or pneumonoultramicroscopicsilicovolcanoconiosis.[24] As discussed above, there is essentially no risk of silicosis developing in the general population simply because exposures have been too low and the exposure time much too short. Even in the occupational groups having the heaviest exposure, such as the loggers salvaging the timber on the mountain, the dust levels are almost certainly considerably lower now than they were in 1980 because the winter rains and snow in 1980 and 1981 compacted the surface of the ash into a crust, which makes it less likely to resuspend when disturbed by wind or humans.

These speculations must be tested scientifically, however. Several groups have now addressed the

question of the potential fibrogenicity of the dust using a variety of studies in animals and in vitro assays. Fruchter and associates[6] were the first to report in vitro biological tests of the ash. These investigators addressed the question of the toxicity of the ash to alveolar macrophages using lavaged macrophages from New Zealand white rabbits, incubated for 22 to 24 hours with volcanic ash, and using polyvinyl-toluene beads as an inert control and α-quartz as a highly toxic (positive) control. The toxicity of each material to alveolar macrophages was then estimated by counting the viable and nonviable macrophages. They found that the ash samples from a number of sites did not differ significantly in cytotoxicity from the inert control but did differ very appreciably and significantly from the quartz control. They predicted from this study that the ash is not highly fibrogenic. Very similar findings using lavaged human macrophages have been reported by Martin and co-workers.[25,26]

A similar conclusion was drawn by Beck and colleagues[27] who used intratracheal instillation of ash in varying concentrations in hamsters with α-quartz used as the fibrogenic control and aluminum oxide and saline used as the inert controls. The lung lavage fluid was then analyzed using a number of different biologic assays that reflect different aspects of lung injury, such as the inflammatory response and the release of cytoplasmic and lysosomal enzymes. They found that the response of the volcanic ash was certainly greater than that of the saline control but was comparable to the response to aluminum oxide, which is generally considered to be relatively inert. The response to α-quartz, on the other hand, was significantly greater than the response to the ash or aluminum oxide for most of the assays. From these data, they concluded that the volcanic ash, though not totally lacking in biologic effects, does not have great potential for causing pulmonary damage unless exposure is prolonged.

Other animal studies using the same technique of intratracheal instillation[28] and studies administering the dust by inhalation (T. R. Martin, MD, personal communication, University of Washington School of Medicine, April 1982) have come to a different conclusion, namely, that the volcanic ash may indeed cause a fibrotic reaction in the lungs. Thus, Green and associates[28] instilled 10 mg of volcanic ash intratracheally in rats, using saline controls, and then killed groups of the exposed or control rats at periods varying from one day to six months. They observed an initial inflammatory response and, later, granuloma formation with most of the granulomas containing foreign body-type giant cells, and some showing central necrosis. Six months after the instillation, the granulomas were larger and contained more collagen than seen earlier.

Martin and co-workers, using the inhalation approach, have come up with very similar findings. They exposed groups of rats to volcanic ash, α-quartz or clean air for six hours a day for ten days, using respirable dust concentrations (volcanic ash and α-quartz particles) averaging about 100 mg per cu m, intended to represent a "worst case" exposure. Immediately after the exposure, alveolar septal thickening and damage to type I pneumonocytes developed in the volcanic ash-exposed animals. Six months later, septal thickening was pronounced and collagen deposition was evident by light and electron microscopy, but no granulomas were seen.

Can these apparently conflicting results be reconciled? My interpretation of them would be that they clearly show that the volcanic ash does not have nearly the cytotoxic or fibrogenic potential of α-quartz but it undoubtedly does have the ability to cause lung injury if deposited in sufficient quantities. In this regard, it is worth pointing out that the exposures in the inhalation studies and the dose instilled intratracheally were very high, much greater than any exposures encountered in an occupational setting and orders of magnitude greater than environmental exposures. The question of whether lower doses delivered over a longer period will also cause lung injury must still be answered by appropriate studies in animals and humans.

Other Health Effects

This review has concentrated so far on the effect of the volcanic ash on the lungs. It would be somewhat myopic to assume that this is the only organ at risk, though the intimate contact of the lungs with the environment makes this organ most likely at risk.

Other possible health effects might be found on the eyes and on mental health. So far, there is relatively little information about the effects on the eyes apart from the initial data from the CDC survey that showed that the number of emergency room visits to two hospitals in Yakima, Washington, for eye irritation and abrasion, foreign bodies and conjunctivitis increased considerably for the

first two weeks following the eruption.[7,8] Whether the eye irritation progresses to a more chronic inflammatory condition with continued exposure is not yet known.

Likewise, there is still relatively little information about the impact of the volcanic activity on mental health. This may turn out to be a very important issue because a report by the US Department of Interior's Geological Surveys warns of possible further dangerous eruptions and heavy ashfalls[7] and has emphasized that other Cascade Range volcanoes may well erupt in the near future. Indeed, the psychosocial problems that stem from living in the shadow of an active volcano may well turn out to be of greater importance than any effect of the ash on the lungs. Extrapolating from data obtained from other natural disasters, especially those that may persist for prolonged periods or have a potential for frequent recurrences, one might predict that feelings of depression, helplessness and loss of control will be common and that varying amounts of stress, bad dreams, sleep problems, guilt, anxiety and avoidance will be seen.

Looking to the Future

The awakening of Mount St Helens in the spring of 1980 has provided scientists of many persuasions the opportunity to study the behavior of volcanoes and the human response to volcanic activity. Despite the existence and activity of volcanoes throughout the world since the beginning of time, there is relatively little solid scientific data relating to the effect of volcanic activity on health. This question assumes considerable importance for those living in the western United States with the realization that a number of the Cascade Range volcanoes are still active and that a sizable population mass is potentially at risk.

We certainly know more about the health effects of volcanic ash than we did in May 1980, but we still do not know nearly enough to be able to give reasoned advice based on solid scientific evidence to public health officials. The advice given at the time of the ashfalls is still appropriate, namely, to minimize exposure to ash by staying indoors when feasible and by using masks approved by the National Institute of Occupational Safety and Health when out in the ash.[7] These have proved to be remarkably effective in reducing the dose of inhaled ash. Deposition of the ash in the lungs can also be minimized by nose breathing. Conversely, the deposition in the lungs will be maximized by exercising vigorously while stirring up the ash and mouth breathing. Jogging and other forms of vigorous outdoor sports should therefore be avoided during and following ashfalls. Outdoor workers who are constantly exposed to the ash should wear adequate respiratory protection and goggles if eye irritation is a problem. Contact lenses should not be worn when dust levels are high.

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